V. EXPOSURE

A general expression for the dose of an individual is the integral over time of the volume of air or water or other contaminated material taken in per unit time, Q, the fraction of the material inhaled or ingested that reaches the blood, n, and the concentration of the material in the air or water or other material, c:

$$D = \int_{t_1}^{t_2} (nQc)dt \qquad (4)$$

Often risk is put in terms of the mean concentration experiences during a lifetime, which would be the integral of Qc divided by the total flow the integral of Q.

The risk estimates given in Section VI make various assumptions about tap water consumption but include no risk factors for eating contaminated fish, which is irrelevant in this situation. The EPA water quality criteria for carcinogens and mutagens are based upon an assumption of 2 liters (L) per day water consumption by the typical 70 kilogram (kg) person (Federal Register, 28 November 1980). Zuilhuis (1982) reports that infants ingest 0.8 L per day or 200-300 ml per kg of body weight, in contrast to the 2 L per day or 3 ml per kg of body weight for adults. He cited a study done in the Netherlands that found that 5% of the men drank more than 2.3 L per day and 5% of the women drank more than 2.1 L per day of water, with 10% indicating they

never drank tap water. Zuilhuis reported that a 1978 study in the United Kingdom found an average intake of 1.26 L per day for adults and and that a study of consumption in the U.S. found 1.63 L per day.

The number of people assumed exposed, N, will change as a result of population migration as well as births and deaths. The rate of change can be described by:

$$dN/dt = (a'+a)N - (b'+b)N \quad (5)$$

where a' and a are birth and immigration rates and b' and b are death and emigration rates, respectively. We have assumed no change in N over time.

The Assabet wells No. 1 and No. 2 supplied about 40% of the water for the 20,000 residents, before the wells were shut off. Water from the Assabet wells was fed into a central distribution system where it was mixed with water from the other town wells (which supplied the remaining 60% of the town's water). This well-mixed water was then distributed to the 20,000 customers. To account for this dilution or mixing effect, the various concentrations predicted at the two wells had to be multiplied by 0.40 to obtain more accurate exposure concentrations. We assumed that concentrations at the tap would not be affected by transport or treatment within the system.

We estimate that only about 1% of the water in the wells is used for drinking. This estimate was derived by comparing the daily intake of the 20,000 users (16000 liters per day or 40% of

40,000 liters) with the total water flow in the two wells, which was typically 330,000 gallons (1.2 million liters) per day for Assabet No. 1 and 230,000 gallons (0.9 million liters) per day for Assabet No. 2 (see Goldberg et al., 1980).

The Assabet River, into which water flows from the study area, empties into the Concord River, which supplies drinking water to the town of Billerica. Therefore, it is necessary to consider the risk to people downstream of the company site.

The Billerica Water Department estimates that about 3.92 million gallons per day (mgd) are drawn from the Concord River, supplying water to about 10,000 families. The flow from the Assabet wells No. 1 and No. 2 was about 0.5 mgd when they were operating. The mean flow of the Assabet River is 120 mgd, based on readings taken by the Army Corps of Engineers. If we assume that all of the water that was withdrawn from the two wells eventually empties back into the river, then the concentrations in the Assabet River should be 0.5/120 or 1/240 times the concentrations in the wells. We may be overestimating concentrations in the river because it is likely that not all of the well water was disposed of in the river. However,we have not included the impact of contamination from the secondary lagoon or landfill on the river.

Before water is withdrawn for consumption by Billerica residents, further dilution takes place because the Assabet River flows into the Concord River. The Concord River has a mean flow rate of 400 mgd, which is about 3.4 times that of the Assabet River. Therefore, the concentration levels of contaminants in the

Billerica water supply would be about 1/800th of those measured at the Assabet wells. The contribution to risk of this path is much less than the direct use of the town wells.

Appendix E is a discussion of the health risks associated with air pollution from the wastewater lagoons, and concludes that such risks are negligible.

VI. RISK ESTIMATION

The equation we use to estimate fatalities requires an estimate of the lifetime risk resulting from having 1 ppb of a given chemical species in the drinking water of a population. Table F-1 in Appendix F gives a summary of such estimates. These risk estimates or risk coefficients were obtained by extrapolating from animal studies on a weight-to-weight basis (Crouch and Wilson, 1979); Clement Associates, 1982), or by using a multistage risk model and extrapolating on a surface-area-to-surface-area basis (EPA's Ambient Water Quality Criteria, EPA's Cancer Assessment Group's estimates, the Council on Environmental Quality's estimates). In Appendix F, we explain how these risk estimates are derived and how we have used them.

Figure 7 presents the range of the estimates of the risk per person per ppb (R) of the indicated chemical in drinking water for a person's 70-year life span. These upper and lower values of the risk estimates were obtained from the sources we selected as authoritative. (The chemical 1,1,1 trichloroethane is not listed because its risk estimate is 5×10^{-10} per person per ppb, too low to be included.)

Two kinds of risk estimates are given in what follows One kind is based on only 10 years of consumption of the water from the town wells, but gives the expected fatalities over a lifetime, 70 years, by averaging the ten years of high concentration with 60 years of assumed zero concentration. The other kind of risk

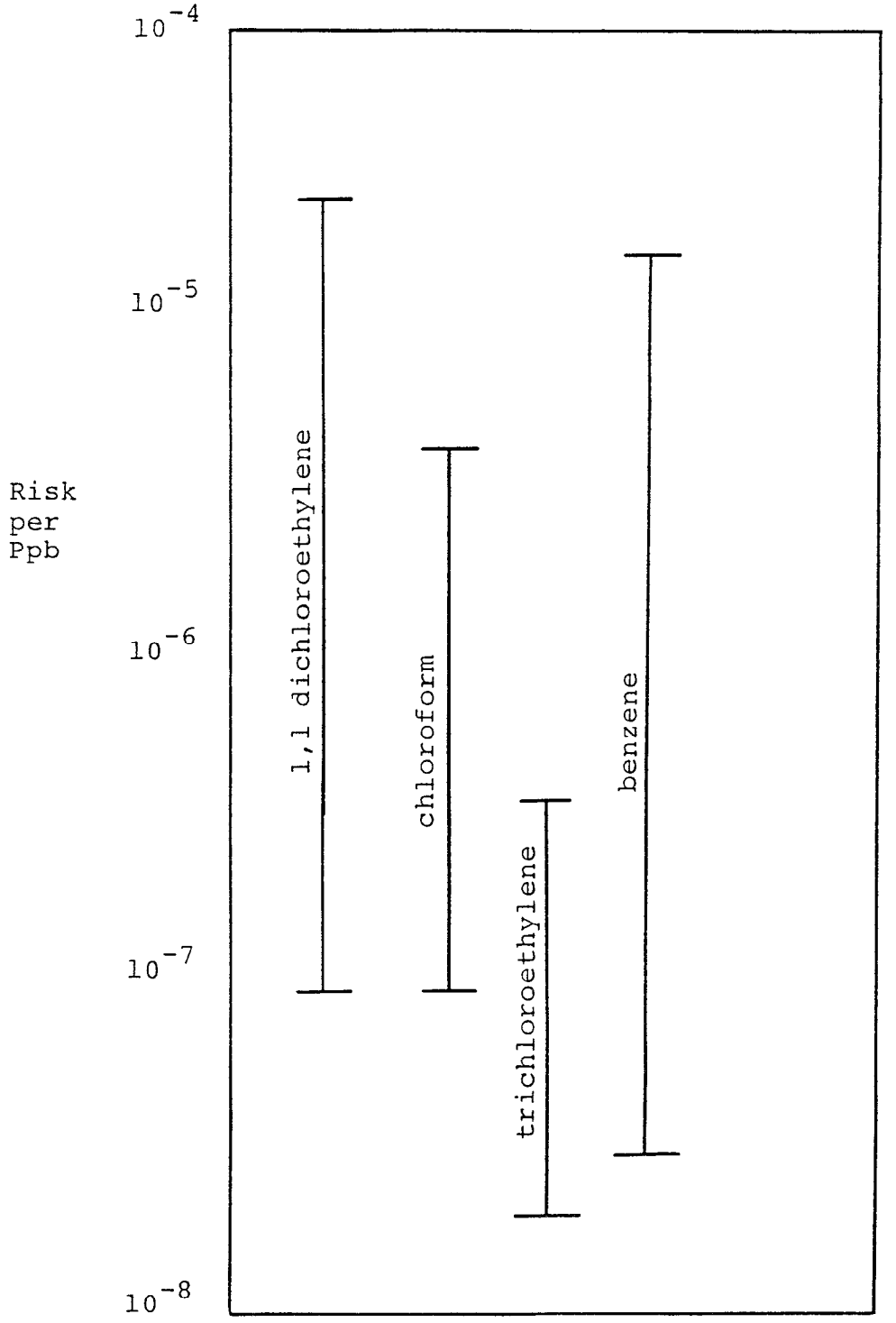

Figure 7. Range of Risk Estimates from Selected Sources, in Terms of Probability of Death per Ppb of Lifetime Average Concentrations in Drinking Water, for Chemicals of Interest

estimate assumes that the wells are used throughout the lifetime of the typical resident (70 years) and that the operations of the company continue throughout that period as well. These estimates are labeled "hypothetical" to emphasize that in the situation studied, action was taken to limit consumption after ten years. These hypothetical values are obtained for two different assumptions: 1. that the levels in 1979 used for the ten-year estimates are also appropriate for the 70-year estimates because equilibrium concentrations had essentially been achieved ("fast transport"); 2. that the levels would rise appreciably over time ("slow transport") because of retention (initially) of the contaminants by the ground, the amount of material released during the twenty-five years of company operations being estimated from the amount of material already in the aquifer inferred from concentrations measured from test borings (which themselves might have failed to capture the contaminants adsorped and absorbed into the soil).

Table 3 gives our estimates of the number of fatalities resulting from exposure to the indicated species; these estimates are based on the assumption that the concentrations found in 1979 in the Assabet wells No. 1 and No. 2 were reached rapidly after the wells were started ("fast transport"). The assumed exposure concentrations are 0.40 times the concentrations of the Assabet well water; 0.40 accounts for the mixing of the Assabet well water with other town water. The concentrations for the two wells were estimated as weighted sums of the typical concentrations for each well, using well pumping rates as weights (60% Assabet No. 1 and

Table 3. Estimates of Hypothetical Fatalities: Wells Used 1970-1979 Only, Fast Transport

Chemical	Concentration measured[d] (ppb)		Assumed exposure concentration[f]	Risk estimates	Estimated fatalities[g]
	A1	A2			
1,1 dichloro-ethylene[a,b]	<10	1-56	8	$3x10^{-5}$ $1x10^{-7}$	0.7 0.002
methylene chloride[b]	nd[e]	Text	8	–	–
chloroform[b]	nd	≤1	0.40	$5x10^{-6}$ $1x10^{-7}$	0.006 0.0001
trichloro-ethylene[b]	1-10	<10	2	$4x10^{-7}$ $2x10^{-8}$	0.002 0.0001
ethylbenzene[b]	1-10	1-10	2	–	–
benzene[c]	nd	≤10	0.8	$2x10^{-5}$ $3x10^{-8}$	0.04 0.00007
chlorobenzene[c]	nd	< 0	0.8	–	–
toluene[c]	nd	<10	0.8	–	–
1,1,1, trichloro-ethane	1-10	<10	2	$5x10^{-10}$	0.000003

a. Both CDM and GZA reports attributed this to the primary and emergency wastewater lagoons.
b. GZA attributed these to lagoons.
c. Not found in lagoons, but nearby.
d. See Goldberg et al., 1980.
e. None detected.
f. Approximately 0.40 [0.6(A1 max/2) + (A2 max/2)].
g. Within 70 years, for the population of 20,000 exposed.

40% Assabet No. 2). Typical concentrations were taken to equal half the upper limit of the concentrations measured in the wells in 1979. The risk estimate appropriate for 10 years of exposure, it is assumed that the well contamination problem would have been found and dealt with when it was discovered in 1979. The risk estimate appropriate for 10 years of consumption equals 10/70th of the lifetime risk estimate. In the case of ten years of exposure, it is assumed that the well contamination problem would have been found and dealt with when it was discovered in 1979. Two risk estimates are given, using the largest and the smallest risk coefficients (taken from Table F-1). The greatest contributor to risk is 1,1 dichloroethylene, if the upper estimate of its risk coefficient is correct. If the lower estimate for 1,1 dichloroethylene is correct, the largest contributor to risk is benzene (using the upper risk coefficient for benzene).

If the fast-transport assumption is correct and the wells had nearly reached equilibrium, the hypothetical fatalities expected over 70 years of operation of the company facilities would simply be those from 1970 to 2040 instead of from 1970 to 1979, or 70/10 times the estimates in Table 3, assuming that the wells are still used over this entire period (see Table 4). In this case, the upper estimate of hypothetical fatalities increases from 0.7 to 5 persons.

Table 5 presents our estimates of the hypothetical number of fatalities caused by the chemicals listed, at the concentrations indicated in the third column. These concentrations are derived from our estimates of the amounts of the particular compounds existing in the plumes in 1979. The final column gives the

Table 4. Estimates of Hypothetical Fatalities: 70 Years' Production/Consumption, Fast Transport

Chemical	Concentration (ppb)	Hypothetical fatalities expected, F
1,1 dichloroethylene	8	4.8, 0.016
methylene chloride	8	
chloroform	0.40	$0.04, 8x10^{-4}$
trichloroethylene	2	$0.016, 8x10^{-4}$
ethylbenzene	2	
benzene	0.8	$0.32, 5x10^{-4}$
chlorobenzene	0.8	
toluene	0.8	
1,1,1 trichloroethane	2	2x10-5

Table 5. Estimates of Hypothetical Fatalities: 70 Years, Production/Consumption Slow Transport

Chemical	Mass assumed[f] (kg)	Concentrations[d] (ppb,) , c	F[g]
1,1 dichloroethylene[a,b]	16,800 1,680	136 13.6	82, 0.3 8, 0.03
methylene chloride[b]	11,200 1,120	88 8.8	– –
chloroform[b]	5,600 560	44 4.4	4, 0.1 0.4, 0.01
trichloroethylene[b]	280 28	2.4 0.2	0.02, 0.001 0.002, 0.0001
ethylbenzene[c]	2,800 280	22.4 2.2	– –
chlorobenzene[c]	– –	– –	– –
toluene[c]	5,600 560	44.8 4.5	– –
1,1,1 trichloroethane[c]	280 28	2.4 0.2	$2x10^{-5}$ 2x10-6
benzene[c]	2,800 280	22.4 2.2	9, 0.01 0.9, 0.001

a,b,c. See comments in Table 3.
d. Concentration = 0.40 (mass (mg) / $(5x10^{7}m^{3})$)
e. Volume = $(2x10^{3}$ m^{3}/day) (70 yr) (365 days/yr)
f. Mass = (70/25) (25-yr estimated mass)
g. F = 20,000 c (R high, R low)

expected fatalities among the 20,000 persons exposed to a lifetime (70 years) of drinking water with these concentrations. Two values are given, corresponding to the highest and lowest risk coefficient per person per ppb, as taken from the sources we considered authoritative. Under the slow-transport assumption, our estimates are based on the ratio of emission rate to well flow rate.

The reader is referred to Appendix D for further discussion of the merits of the fast transport model as compared with the slow transport model. Fast transport is shown to be somewhat more likely than slow transport.

The potential risk to Billerica residents can also be estimated using Eq. 1. Although the number of persons in Billerica using Assabet water (10,000 households or 40,000 persons for families of an average size of 4) is about twice the number of Acton users, the concentrations in Billerica water are expected to be less than 1/800th of the concentrations at the Assabet wells. Applying Eq. 1 to the Billerica case we see that:

$$F' = (2N)\left(\frac{1}{800}\bar{c}\right)R \qquad (6)$$

$$F' = 0.0025(N\bar{c}R) = 0.0025F$$

For the F expected fatalities in Acton, the corresponding number of expected fatalities in Billerica is equal to the product of 0.0025 and F_o.

Tables 6 through 9 present estimates of the number of expected fatalities associated with exposure to various combinations of chemicals: we assume that the effects of multiple exposures are additive. Table 6 presents the set of estimates we have made of

Table 6. Set of Hypothetical Fatality Estimates: Wells Used 10 Years Only, Fast Transport

Chemical (low = 1 or high value = 2 risk estimate used)									Expected fatalities
DCE	MC[a]	C	TCE	EB[a]	B	CB[a]	T[a]	111-TCE[b]	
1		1	1		1			1	0.0025
1		1	1		2			1	0.0473
1		1	2		1			1	0.0047
1		1	2		2			1	0.0494
1		2	1		1			1	0.0080
1		2	1		2			1	0.052
1		2	2		1			1	0.010
1		2	2		2			1	0.054
2		1	1		1			1	0.68
2		1	1		2			1	0.73
2		1	2		1			1	0.68
2		1	2		2			1	0.73
2		2	1		1			1	0.69
2		2	1		2			1	0.73
2		2	2		1			1	0.69
2		2	2		2			1	0.73

a. Risk estimates not available for these chemicals.
b. Only one risk estimate has been made for 1,1,1-trichloroethane.

Table 7. Set of Hypothetical Fatality Estimates: 70 Years' Production/Consumption, Fast Transport

Chemical (low = 1 or high value = 2 risk estimate used)								Expected fatalities
DCE	MC[a]	C	TCE	EB[a]	B	CB[a]	T[a]	111-TCE[b]	
1		1	1		1			1	0.018
1		1	1		2			1	0.33
1		1	2		1			1	0.033
1		1	2		2			1	0.35
1		2	1		1			1	0.057
1		2	1		2			1	0.37
1		2	2		1			1	0.072
1		2	2		2			1	0.39
2		1	1		1			1	4.8
2		1	1		2			1	5.1
2		1	2		1			1	4.8
2		1	2		2			1	5.1
2		2	1		1			1	4.8
2		2	1		2			1	5.1
2		2	2		1			1	4.8
2		2	2		2			1	5.1

a. Risk estimates not available for these chemicals.
b. Only one risk estimate has been made for 1,1,1-trichloroethane.

Table 8. Set of Hypothetical Fatality Estimates: 70 Years' Production/Consumption, Lower Estimate of Emission Rates, Slow Transport

Chemical (low = 1 or high value = 2 risk estimate used)									Expected fatalities
DCE	MC[a]	C	TCE	EB[a]	B	CB[a]	T[a]	111-TCE[b]	
1		1	1	1				1	0.03
1		1	1	2				1	0.93
1		1	2	1				1	0.03
1		1	2	2				1	0.93
1		2	1	1				1	0.46
1		2	1	2				1	1.36
1		2	2	1				1	0.47
1		2	2	2				1	1.3
2		1	1	1				1	8.1
2		1	1	2				1	9.0
2		1	2	1				1	8.1
2		1	2	2				1	9.0
2		2	1	1				1	8.6
2		2	1	2				1	9.4
2		2	2	1				1	8.6
2		2	2	2				1	9.4

a. Risk estimates not available for these chemicals.
b. Only one risk estimate has been made for 1,1,1-trichloroethane.

Table 9. Set of Hypothetical Fatality Estimates: 70 Years' Production/Consumption, Upper Estimate of Emission Rates, Slow Transport

Chemical (low = 1 or high value = 2 risk estimate used)									Expected fatalities
DCE	MC[a]	C	TCE	EB[a]	B	CB"	T[a]	111-TCE[b]	
1		1	1		1			1	0.37
1		1	1		2			1	9.32
1		1	2		1			1	0.39
1		1	2		2			1	9.3
1		2	1		1			1	4.6
1		2	1		2			1	13
1		2	2		1			1	4.7
1		2	2		2			1	13
2		1	1		1			1	81
2		1	1		2			1	90
2		1	2		1			1	81
2		1	2		2			1	90
2		2	1		1			1	86
2		2	1		2			1	94
2		2	2		1			1	86
2		2	2		2			1	94

a. Risk estimates not available for these chemicals.
b. Only one risk estimate has been made for 1,1,1-trichloroethane.

expected fatalities from our fast transport model, assuming 10 years of well use. The column marked "Expected Fatalities" is the number of fatalities given particular assumptions about the risk coefficients. These assumptions are shown in the first nine columns, where "1" denotes the use of the low end of the estimate range and "2" denotes the use of the high end of the range. The chemicals are given in the following order: 1,1, dichloroethylene (DCE), methylene chloride (MC), chloroform (C), trichloroethylene (TCE) ethylbenzene (EB), benzene (B), chlorobenzene (CB), toluene (T), and 1,1,1 trichloroethane (TCE). The chemicals for which we could not get authoritative risk estimates (MC, EB, CB, T) do not contribute to the fatality estimates.

Hypothetical fatality estimates for 70 years of production and consumption under the fast transport assumption are shown in Table 7 and can be obtained by multiplying the values for expected fatalities in Table 6 by 70/10=7.

Table 8 gives a similar summary for the case in which the operations and exposures continue for 70 years and slow transport is assumed. The slow transport assumption has been implemented with two estimates of mass in the aquifer, a factor of ten apart, leading to a factor of ten difference in the assumed mean concentrations over a lifetime. Table 8 uses the lower emission rate estimates and Table 9 uses the higher emission rate estimates for 70 years. Values in the tables can be adjusted for a period of emissions shorter than 70 years by multiplying the expected fatality values by the ratio of the period of interest to 70 years.

Table 10 shows our estimates of the expected number of illnesses resulting from the indicated types of toxicity for the exposed population of 20,000, assuming a ten-year exposure. The first column identifies the chemical; the second, the assumed concentration in the Acton drinking water; the third through the eighth show the number of illnesses of various types. The values of risk per person per ppb of chemical in the drinking water are based on the work by Clements Associates, and these values are presented and discussed in Appendix F. Table 11 gives our estimates for the hypothetical number of illnesses expected for the 20,000 people exposed, assuming a 70-year exposure (lifetime) and assuming that transport is slow. The columns are analogous to those in Table 10.

The following factors add uncertainty to our fatality and illness estimates:

1. Whether the contamination would have been found when it was and the wells closed. The time of discovery (hence, the length of exposure) makes a difference of a factor of five or more in the number of expected fatalities.

2. The average mass rate of emission to be expected over the period is questionable. The rate of emission seems to be uncertain by about a factor of ten. The uncertainty could be even greater because the techniques used to obtain the concentrations did not capture and measure the material bound up in the soil.

Table 10. Estimate of Hypothetical Illnesses: Wells used 1970-1979 Only Fast Transport

Chemical	Assumed Conc. (ppb)	Estimate of Number of Cases Due to:[a] Reproductive Toxicity	Terato-genicity	Hepato-toxicity	Renal Toxicity	Neuro-behavioral Toxicity	Other Toxicity
1,1-dichloroethylene	8	–	–	.004	-	–	–
methylene chloride	8	–	–	.0003	.0007	.002	.0003
chloroform	0.40	.0001	.0001	.001	.001	.0003	.0003
trichloroethlyene	2	–	–	.00009	.00003	.02	.03
ethylbenzene	2	–	–		–		
benzene	0.8	.0004	.00003	-	–	.0005	.0003
chlorobenzene	0.8	–	–	–	–	–	–
toluene	0.8	.00002	–	.000008	-	.0007	
1,1,1-trichloroethane	2	.000009	-	.0001	-	.0001	.00006

[a] The risk coefficients (R) for the various tyupes of illnesses are taken from Table F-2. The population at risk is 20,000.

Table 11. Estimate of Hypothetical Illnesses: 70 Years' Production/Consumption, Upper & Lower Emmission Rate Estimates Slow Transport

Chemical	Assumed Conc. (ppb)	Estimate of Number of Cases Due to:[c] Reproductive Toxicity	Terato-genicity	Hepato-toxicity	Renal Toxicity	Neuro-behavioral Toxicity	Other Toxicity
1,1-dichloroethylene	136[a]	–	–	.430	–	–	–
	13.6b	–	–	.043	–	–	–
methylene chloride	88	–	–	.021	.055	.181	.019
	8.8	–	–	.002	.005	.018	.002
chloroform	44	.103	.087	.816	.818	.020	.239
	4.4	.010	.009	.082	.082	.002	.024
trichloroethlyene	2.4	–	–	.0007	.0003	.129	.213
	0.2			.00007	.00003	.013	.021
ethylbenzene	22.4	–	–	–	–	–	–
	2.2	–	–	–	–	–	–
benzene	22.4	.079	.006	–	–	.097	.057
	2.2	.008	.0006	–	–	.010	.006
chlorobenzene	–	–	–	–	–	–	–
	–	–	–	–	–	–	–
toluene	44.8	.007	–	.003	–	.290	–
	4.5	.0007	–	.0003	–	.030	–
1,1,1-trichloroethane	2.4	.00008	–	.001	–	.001	.0005
	0.2	.000008	–	.0001	–	.0001	.00005

[a] Estimated Upper Emission Rate
[b] Estimated Lower Emission Rate
[c] The risk coefficients (R) for the various types of illnesses are taken from Table F-2. The population at risk is 20,000.

3. The risk estimates resulting from a lifetime's ingestion of the chemical species at 1 ppb concentration differ by factors of 300 (1,1 dichloroethylene), 50 (chloroform), 20 (trichloroethylene), and 700 (benzene). Several of the chemicals had no risks estimates made by the sources we selected.

4. A host of many factors that we believe contribute less uncertainty than those listed above.

VII. VALUATION OF BENEFITS OF CONTROL

Although much has been said and written about valuing life, we are not going to recapitulate the arguments here. The estimates and hypothetical estimates of fatalities in Tables 3, 4, and 5 can be made into cost estimates by multiplying the expected number of fatalities by the values of life typically used for regulatory analysis -- $330,000 to $2,500,000 (in 1980 dollars). We also note that the value of life extension or the prevention of death may properly be discounted to obtain present values from future values.

The ratio of the two estimates of the value of life extension ($/F) is about 8:1, and the ratio of the estimates of the toxicity of DCE is 300:1. Referring to Table 6, the expected costs of the ten-year exposure are dominated by the choices of the DCE risk coefficient and the choice of $/F. Roughly one-quarter of the combinations would have both of these variables at their highest values and would give estimated costs near $1.8 million. Another quarter would have the risk coefficient for DCE at its highest value, but $/F at the lowest value, giving cost estimates near $0.2 million. The lower risk estimate for DCE and the higher $/F value give estimates near $0.1 million. The lowest quartile, with the lower $/F value and the lower risk coefficient for DCE, is near $5000. Similar cost estimates can be made for Tables 7, 8 and 9.

VIII. LIMITATIONS

Risk estimation as performed above is more objective than expert opinion on whether a situation is "hazardous"; it may well be more accurate, but it certainly has its limitations. Knowledge of the emission rates and even of the species emitted is quite uncertain: some species may be present but undetected; some contamination attributed to the lagoons may have other sources; areas not sampled may contain more or less material than assumed. The transport model makes no allowance for chemical transformations, for either the destruction or creation of toxic species. The concentrations as measured were often near the limits of detection, making estimation difficult. The dose/response relationships are likely to be artificially high if linear approximation at low doses is incorrect. The Cancer Assessment Group's estimates contain an upward bias because of the selection of the 95th percentile estimate for extrapolation back to zero, although other factors (such as errors in the independent variables used in regressions) can bias the estimates downward as well. Neither synergistic nor antagonistic relationships between chemicals are considered.

Valuation is, on the one hand, an attempt to estimate the costs of saving lives, but on the other, it is an attempt to estimate the cost to society of a life lost. In making decisions, it is often appropriate to include a consideration of voluntary versus involuntary risks and careful versus negligent behavior, neither of which is reflected in cost estimates.

Still, even with such limitations, risk estimation is an appropriate part of our continuing efforts to understand our environment and act upon that understanding. Because of the uncertainties noted above, our risk estimates cover a very wide range. Such risk estimates would be most helpful where that range is clearly above or below a value used as a decision criterion, such as the cost of a proposed method for preventing contamination of drinking water wells.

APPENDIX A
GEOLOGICAL CONSIDERATIONS

S. Pancoast

Introduction

A general knowledge of geological conditions affecting groundwater movement is necessary if the risks associated with solid or liquid wastes disposed of on land are to be estimated properly.

Infiltration of rainfall through the unsaturated zone in the soil can and often does lead to the leaching of various chemical substances from wastewater lagoons or similar unprotected surface impoundments. The degree of infiltration depends largely on soil moisture, soil texture, vegetative cover, land slope, and frost penetration (Caswell, 1979).

Once in the unsaturated zone or the zone of aeration, water typically follows the path of least resistance (see Figure A-1). Knowledge of the water's velocity and direction is essential to determine the quantity of hazardous contaminants dissolved in water at a particular location.

Among the most important properties of sediments and rocks to be considered when discussing groundwater movement are their porosity and permeability. Permeability is a measure of the relative ease with which a porous medium transmits a liquid

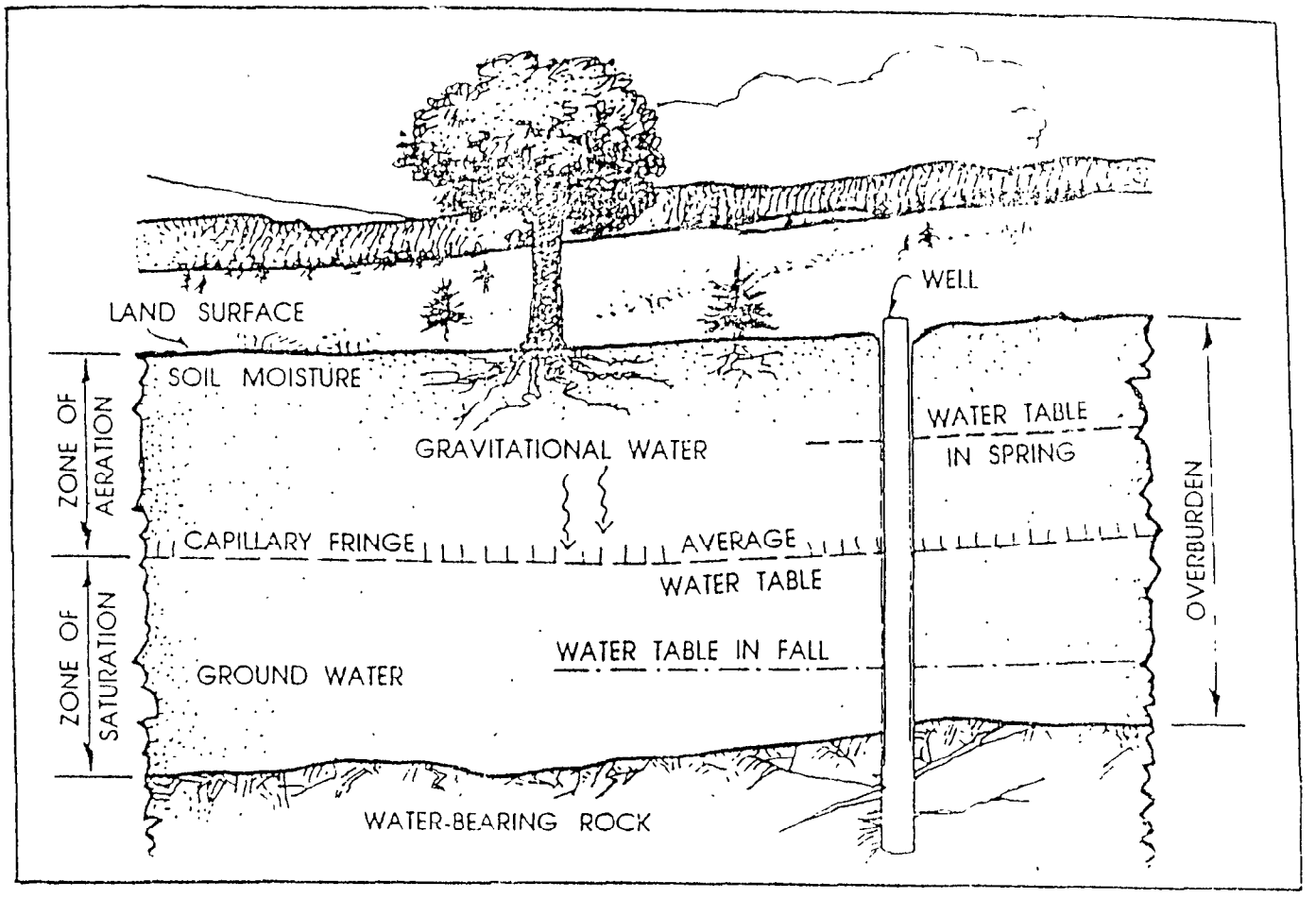

Figure A-1. Schematic of Subsurface Hydrogeology (Caswell, 1979)

(Lycott, 1981). Porosity is the property of a rock or unconsolidated material to contain voids or empty spaces; it may be expressed quantitatively as the ratio of the volume of its open spaces to its total volume (Goldberg et al., 1980).

Monitoring the distance a pollutant travels through a particular geologic deposit depends largely on the chemical makeup of both the subsurface material and the contaminant itself. The same material deposited under different conditions may exhibit very different behavior.

Sinking Pond Aquifer

The geology of the town is best described as a product of glacial action. The last major ice sheet overrode bedrock and deposited glacial till over most of the town, including the southeastern corner where the facilities of the company overlie the Sinking Pond aquifer. In this area, stratified drift (materials sorted by water) was laid down by glacial meltwaters. Stratified drift has a higher permeability than glacial till (also found in many parts of Acton), especially if it consists of coarse grains and gravels (Caswell, 1979).

An aquifer is a geological formation, group of formations, or part of a formation that contains sufficient saturated, permeable materials to yield significant quantifies of water to wells and springs (Caswell, 1979). The Sinking Pond aquifer is one of eight aquifers in the town and until December 1978, it provided 40% of the water for the residents. Most aquifers in New England are constrained by bedrock valley walls and have a stream passing through them; such aquifers are termed "valley

fill". The aquifers in the town are all valley fill, consisting of bedrock valleys that were filled in, to a nearly level condition, by glacial outwash (Caswell, 1979).

The potability of an aquifer depends largely on the characteristics of the soil in the zone of aeration. This zone is where the essential filtering processes take place and where potentially hazardous materials passing through may or may not become neutralized. Aquifers underlying a thick, unsaturated soil cover are better protected than those with little or no overburden.

Uncemented, stratified sediments form the skeleton of the Sinking Pond aquifer and its respective overburden. Central and southeastern sections consist of thick beds of fine sand and some silt.

When estimating the velocity and direction of groundwater movement throughout this area, homogeneity and isotropism of subsurface materials are assumed, before calculations involving permeability and porosity are made. Homogeneity refers to a similarity in texture of the materials through which the water is flowing, while isotropism means that the material is equally permeable in all directions (Caswell, 1979). In the case of Acton, this generalization may be justified after examining a subsurface geologic profile map (see Figure A-2). One can see that all the materials immediately surrounding Sinking Pond range from fine to coarse-grained sand, or 0.0625 to 2.00 millimeters

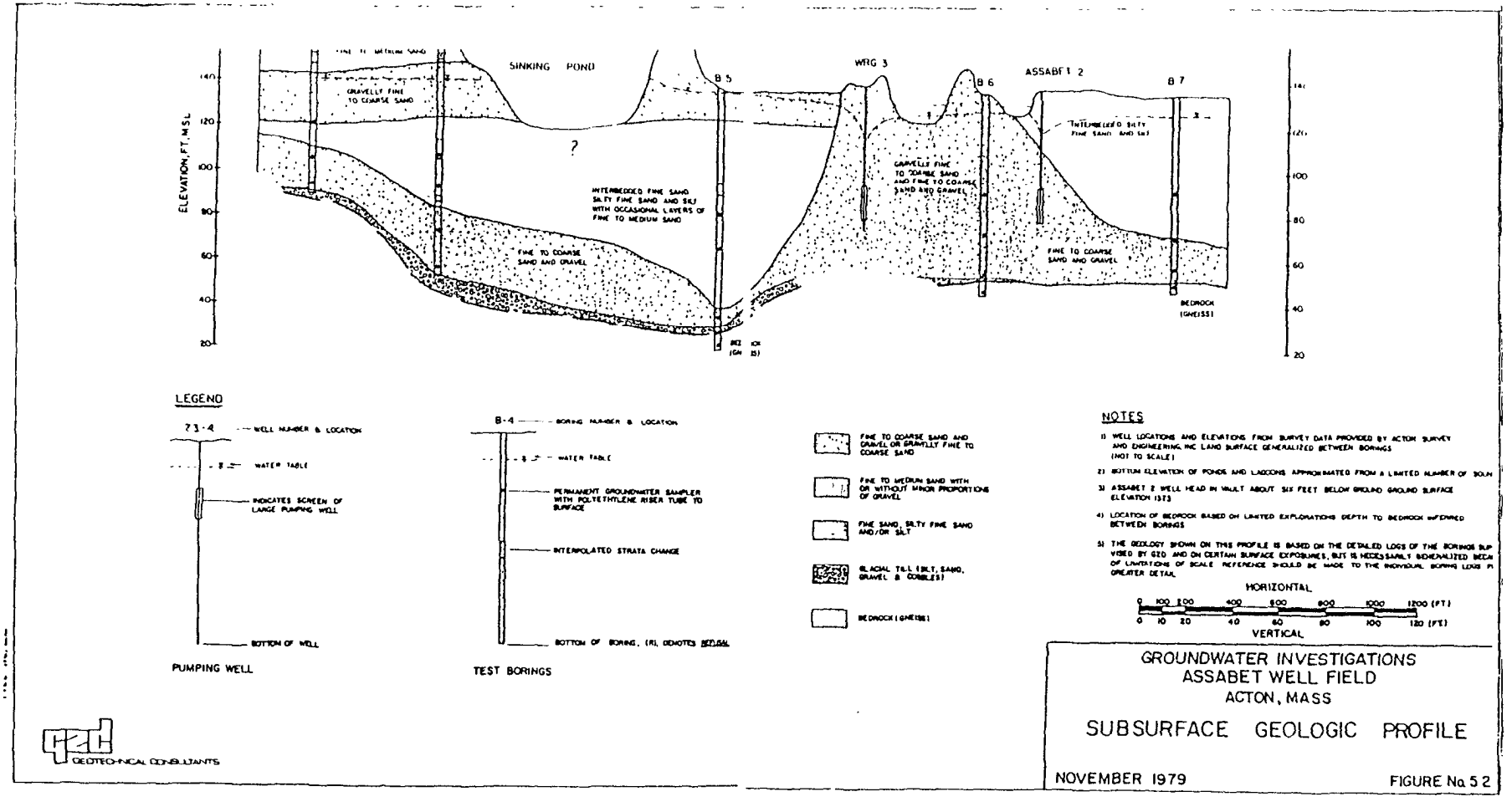

Figure A-2. Profile of Subsurface Geology (Goldberg at al., 1980)

(mm) in diameter. Because silt and clay particles are much smaller, generally being less than 1/300 mm in diameter, groupingthe sands together as one single layer of overburden is not unusual.

Porosities may vary slightly around Sinking Pond since the upper layer is somewhat less porous than the lower layer; however, the permeability of this specific site (for practical applications) is generally the same throughout. The study area is not interrupted by clay lenses or igneous intrusions which could inhibit the passage of water and significantly affect groundwater modeling results.

The two affected municipal wells, which draw from Sinking Pond aquifer, Assabet wells No. 1 and No. 2, are distinctly water table wells. A water table well is essentially an enlarged pore in a porous medium that fills with water to the level of the surrounding water table (Caswell, 1979). Seasonal variations, as well as induced pumping, affect the level of the water table.

When water is pumped out of a water table well, surrounding water immediately begins to slope inward toward the well. In a homogenous and isotropic material (which are two of our primary assumptions), pumping causes a cone of depression to form, with the well bottom at the apex. Flow potential is lower than the surrounding aquifer and thus groundwater flows into the well by gravity (Caswell, 1979). In the Sinking Pond aquifer, the water flowing into these wells comes from the northeast and passes to the southwest where it ultimately empties into the Assabet River.

The more wells that a given body of water encounters, the more distorted the cones of depression become. Therefore, the wells in the Sinking Pond aquifer could play a significant role in the distribution of contaminants -- an important thought to keep in mind when examining the site. Only small drawdowns are encountered generally, but in areas where the aquifer is thin, pumping could induce significant drawdown. In the latter case, the plumes may overlap and further complicate modeling efforts.

APPENDIX B
SOURCE ESTIMATION METHODOLOGY

J.A. Sullivan

Introduction

Waste disposal practices on the company site have not been carefully monitored or documented over the last forty years. Estimates of the amounts of chemicals disposed of on the site are not available. In 1980, the Environmental Protection Agency and the Massachusetts Department of Environmental Quality Engineering required the company to submit a report describing the types and quantities of chemicals disposed of on the site. Unfortunately, the waste disclosure report does not provide a quantitative chemical inventory of the company's activities. Therefore, to estimate the amounts of the contaminants dumped onto the site, we had to draw upon data collected by Goldberg, Zoino and Associates (GZA), consultants who performed a sampling and analysis program for the site.

This appendix describes the methodology used to estimate the amounts of the eight contaminants of interest in the aquifer; these estimates will be used in our slow transport model. The assumptions we have had to make and the limitations of the methodology are also discussed.

Estimation of Chlorinated Hydrocarbons

An estimate of the mass of the following five compounds was made using data collected by GZA in their August 1979 sampling

program: 1,1 dichloroethylene, methylene chloride, trichloroethylene, chloroform, and 1,1,1 trichloroethane. The following calculations represent our best estimate of the amount of contaminant present in the aquifer at the time of sampling.

In the second volume of Goldberg et al., 1980, several contaminant distribution maps are provided. Area1 concentration profiles were mapped for each of the five chlorinated compounds. Both vertical and area1 concentration profiles are provided for total chlorinated hydrocarbons (see Figures B-1 and B-2). The maps of total chlorinated compounds were made by summing up the concentrations of the individual chlorinated species identified in the aquifer. Although other chlorinated compounds were included in the sum, their contribution to the total was negligible. Because the chlorinated compounds were found in higher concentrations along the bottom of the aquifer (they were denser than water), as seen in Figure B-1, we estimated the mass of the individual compounds using GZA's vertical profile for total chlorinated hydrocarbons. The areal distribution maps of the five species were used to calculate the relative weights of the five, as part of the total mass of chlorinated hydrocarbons, and to indicate the horizontal extent of the contaminants.

Figure B-1 represents inferred vertical concentration. gradients along an axis drawn between B1 and B7 in the Sinking Pond aquifer. This line or cross-sectional cut through the aquifer is approximately parallel to the direction of groundwater movement (Goldberg et al., 1980). GZA installed ten multilevel observation wells (B1 through B10) along this axis to study how contaminant levels vary with depth.

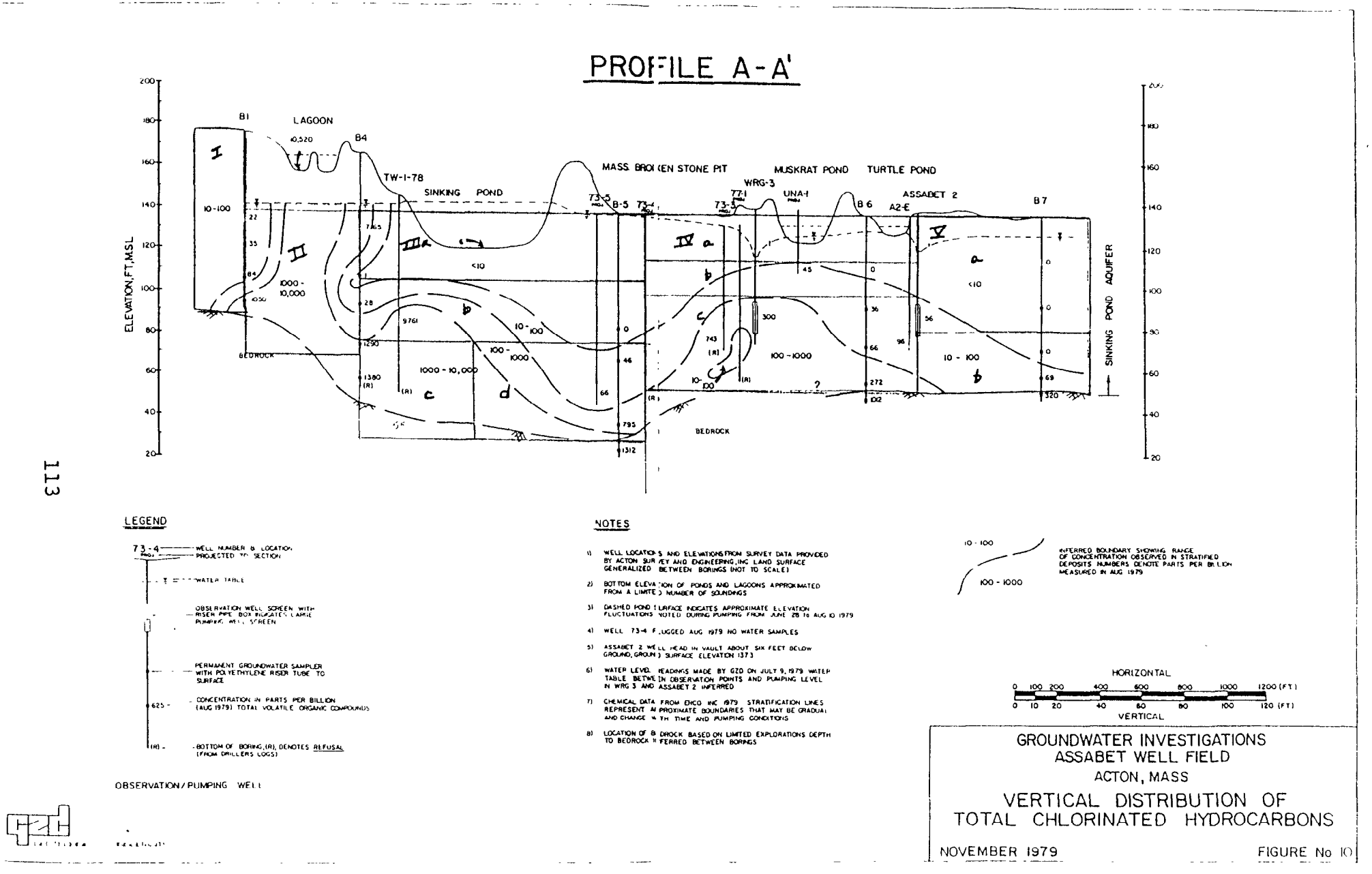

Figure B-1. Vertical Distribution of Total Chlorinated Hydrocarbons (Goldberg et al., 1980) with Superimposed Boundaries, Used for Integrating Concentrations Over the Aquifer Volume

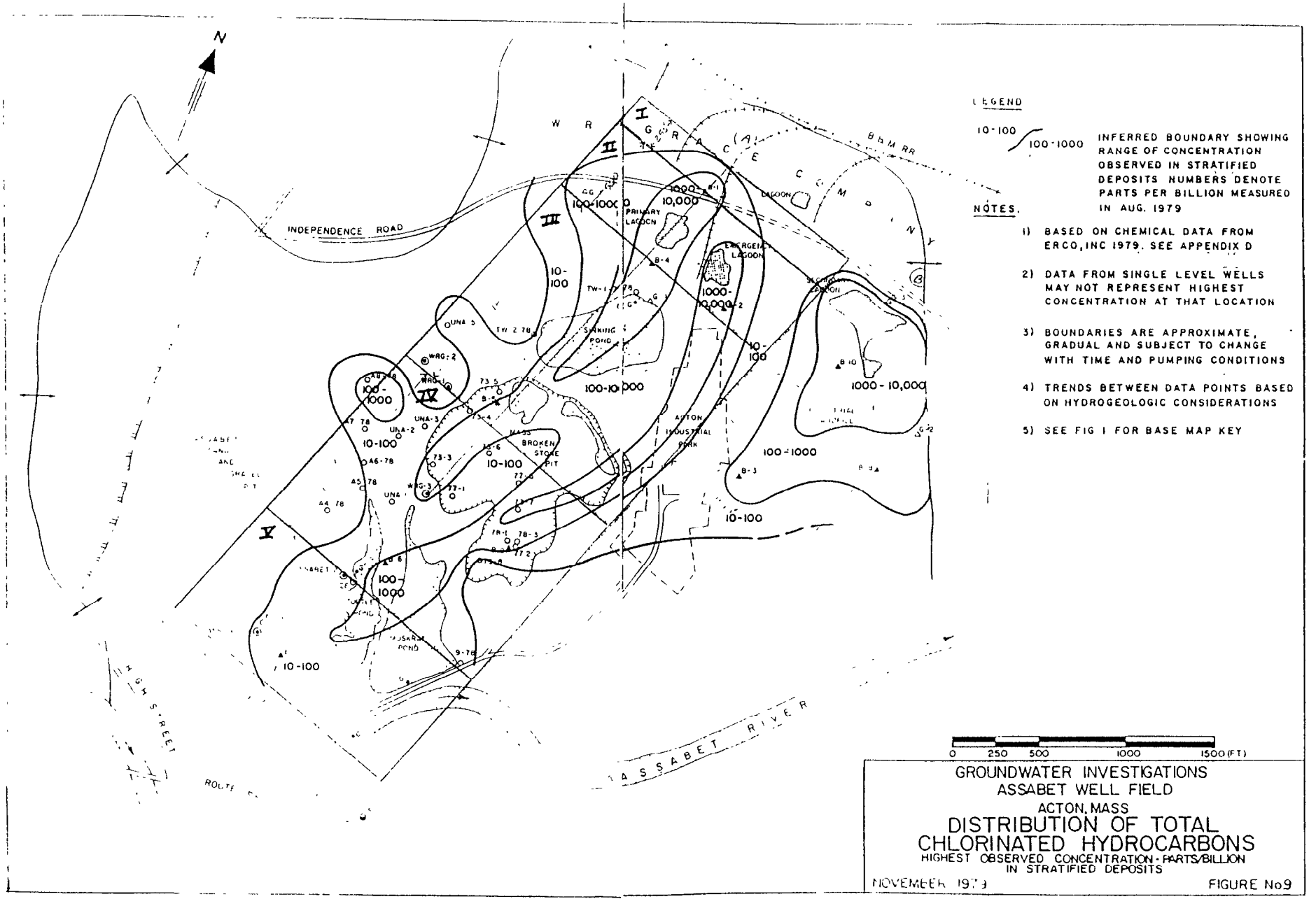

Figure B-2. Horizontal Distribution of Total Chlorinated Hydrocarbons (Goldbert et al., 1980) with Integration Boundaries Superimposed

First we partitioned the slice of the aquifer as shown in Figure B-1 into eleven regions of approximately uniform concentration; each region was assigned a depth using the elevation scale shown in the figure. Then, as seen in Figure B-2, we delineated the boundaries of the contaminant plume and drew in areas to correspond to the regions in Figure B-1. Consequently, the contaminant plume was cut up into compartments of equal concentrations, each possessing a characteristic volume.

The basic equation used to estimate the mass of total chlorinated hydrocarbons present in the aquifer, M (in kilograms), during GZA's sampling period is

$$M = \sum_{i} \sum_{j} p v_{ij} c_{ij} \qquad \text{(B-1)}$$

where:

i = I, II, ..., V (refer to Figures B-1 and B-2)
j = a,b,c,d (refer to Figures B-1 and B-2)

p, the porosity, is assumed to be constant across the aquifer and equal to 0.40. v_{ij} is the volume of the ijth compartment and c_{ij} is the concentration of the ijth compartment. As discussed in the body of this report, we obtained upper and lower emission rate estimates by assigning both the maximum and minimum concentration isopleth limits, which differ by factors of ten, to a given compartment.

The above calculation, which resulted in an estimated range of 1200 to 12,000 kilograms of total chlorinated hydrocarbons emanating from the primary and emergency lagoons, is based on the assumption that the vertical concentration profile in Figure B-1

is constant across the width of the plume. Because concentration levels decrease as one goes farther from the axis, our upper estimate is undoubtedly an overestimate.

The area1 distribution maps of the individual compounds were used to estimate the relative percentage of each compound in the total mass of chlorinated compounds. Again, the plume was partitioned into compartments of equal concentration and equal depth. A volume was obtained for each compartment, assuming a uniform depth to bedrock of 90 feet.

The areal maps do not stratify concentration by depth; therefore, for the relative percentage calculation we assumed uniform vertical concentration profiles. We used these maps solely to predict the relative amounts of the individual compounds. The mass of an individual compound, such as methylene chloride, was based on its percentage of the total mass of chlorinated hydrocarbons compared with the other four compounds and on our total mass calculation (the volume integration).

Estimation of Aromatic Hydrocarbons

GZA did not provide a vertical concentration profile of total aromatic hydrocarbons. To estimate the amount of benzene, toluene, and ethylbenzene present in the plumes originating from the primary lagoon, we constructed a vertical profile (along the same axis) for total aromatic hydrocarbons, in very much the same way GZA did for the chlorinated species. We did not include secondary lagoon plumes nor any contamination close to the Assabet River.

As shown in Figure B-3, we partitioned the plume, an area approximately between wells B1 and B5 (where the plume ends), into compartments centered around the sampling points. An individual compartment was assigned a concentration equal to the sum of the concentrations of benzene, toluene, and ethyl-benzene at that particular well and depth. A volume was calculated for each compartment, and the total mass of aromatic hydrocarbons was then determined. This method provided a point estimate. Because of the uncertainty involved in making source estimates, we sought a range of estimates. To that end, we assumed that the upper and lower estimates of total mass differed by about a factor of 10, which is consistent with the method we used to estimate the amount of chlorinated hydrocarbons. Therefore, we multiplied and divided the point estimate by the square root of 10 to obtain an upper and lower bound on the total mass of aromatic compounds present. And as we did earlier, we estimated the relative fraction of benzene, toluene, and ethylbenzene present using the areal distribution maps that GZA provided for the three compounds.

Total Hydrocarbons

A third estimate of the volume of hydrocarbons present in the interstitial water was made by J.A. Atoeckle, a graduate student at the Harvard School of Public Health (personal communication, 1983). He averaged, region by region, 38 concentration readings obtained within the regions described in

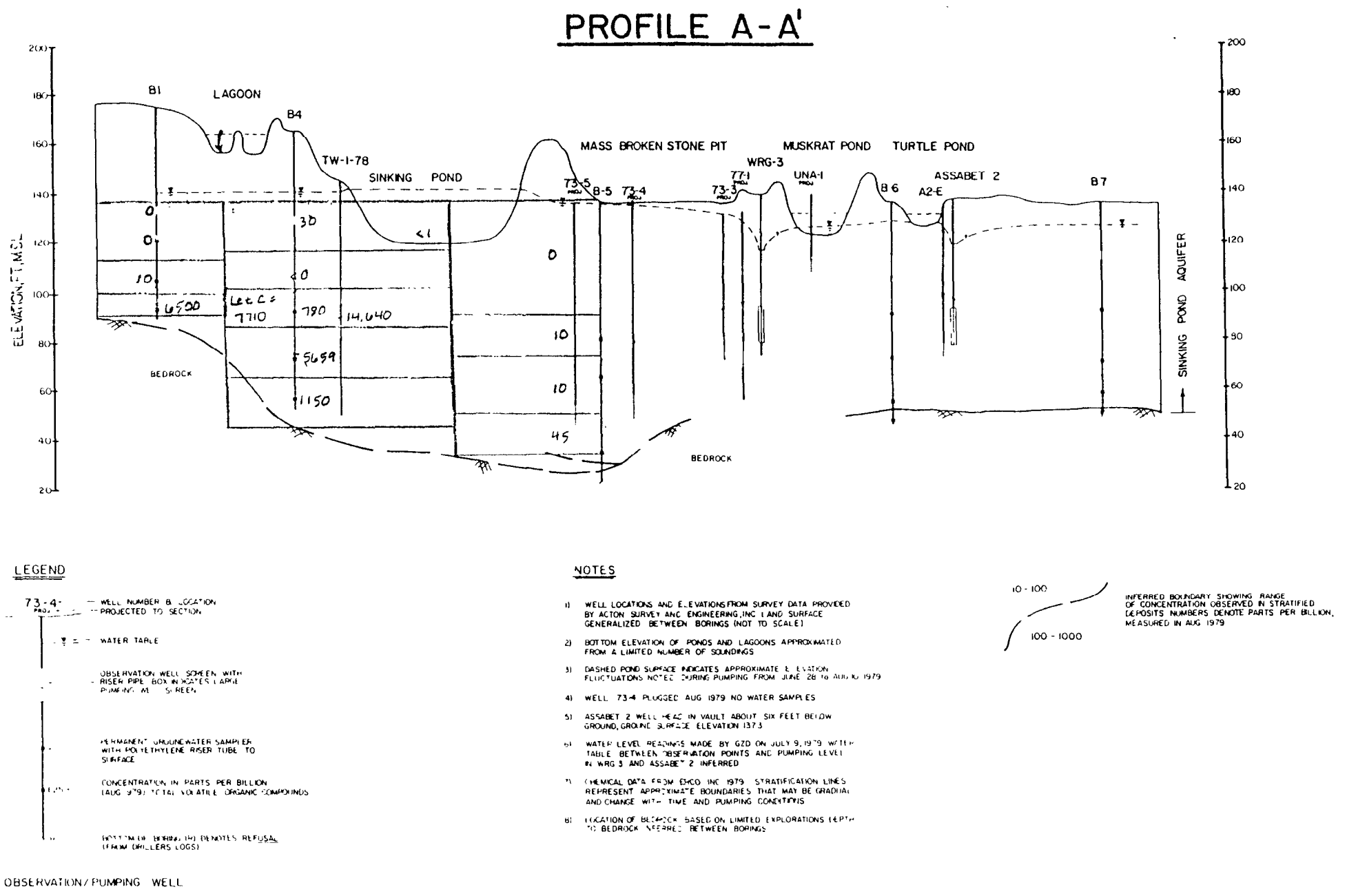

Figure B-3. Vertical Distribution of Benzene, Toluene, and Ethylbenzene, Used for Integrating Concentration Over the Aquifer Volume (Based on Goldberg et al., 1980)

Figures B-1 and B-2. He multiplied the average concentration for a region by the volume of the region and by the porosity. His value for total hydrocarbons was 4×10^3 kg the sum of the products. A summary of his calculations is presented in Table B-1.

Conclusions and Limitations

The above calculations were performed to obtain gross estimates of the amounts of pollutants present in the plumes in August 1979. In the absence of better records of the company's inventories and practices, we relied on data collected by GZA over a ten-day period in 1979. As GZA acknowledges, the levels of contaminants in the aquifer vary with seasonal fluctuations. GZA recommended a second sampling round to verify contaminant trends, before beginning aquifer restoration.

The ranges for our estimates of contaminant mass are large, but necessarily so, in light of the information and data available.

The concentrations used to form the profiles did not include material, if any, that was bound to the soil in the vicinity sampled, and thus we may be systematically underestimating emissions. Soil adsorption would be expected to be important in the case of slow transport, the case we used for the volume integration.

Table B-1. Concentration Volume

Region	(ug/1	(10^6 1) Regional mass (kg)	Number of observations/ region
I	295	214	63	4
II	1831	723	1324	8
III,a	385	896	345	2
III,b	10	702	7	2
III,c	4144	450	1866	3
III,d	593	675	400	4
IV,a	0*	396	0*	0
IV,b	23	396	9	2
IV,c	216	1050	227	6
V,a	19	665	13	4
V,b	337	428	161	3
Total		6595	4415kg	38

*assumed value

APPENDIX C
COMPARISON OF CONSULTANT'S REPORTS ON CONTAMINANT SOURCES

A. D. Schatz

Two consulting firms, Goldberg, Zoino and Associates (GZA) and Camp, Dresser and McKee, Inc. (CDM) were hired by the town and the company, respectively, to determine the probable sources of chemical contamination at the town wells. This section compares the consultants' final reports and presents the case for concluding that the company's waste lagoons are the current or potential source of contamination by six chemicals at the town wells, Assabet No. 1 and No. 2. (See Table C-1).

Of the eight chemicals whose plumes were described by GZA, six appear to have emanated from the company's lagoons. They are benzene, toluene, ethylbenzene, 1,1 dichloroethylene, methylene chloride, and chloroform.

The major source of trichloroethylene contamination at the town wells, according to both CDM and GZA, is induced infiltration from the Assabet River. The company's disposal of this chemical was primarily in the secondary lagoon, from which little or no contamination appears to be migrating toward the wells.

1,1,1 Trichloroethane comprises only about 1% of all contamination found in the aquifer. Although a region of dilute contamination was identified by GZA just north of the Assabet No. 2 well, GZA did not assert a link with the company's disposal

Table C-1. Comparison of GZA and CDM Conclusions

Chemical	Probable sources	
	GZA	CDM
benzene, toluene, and ethylbenzene	lagoons	abandoned fuel storage tank in Massachusetts Broken Stone Pit or fuel storage and filling station at Assabet Sand and Gravel Co.
1,1 dichloroethylene	primary & emergency lagoons	primary & emergency lagoons
methylene chloride	lagoons & river	none detected
chloroform	lagoons	none detected
trichloroethylene	lagoons & river	river
1,1,1 trichloroethane	not identified	river

Other Comments:

By GZA:

1. An abandoned underground fuel storage tank in the Massachusetts Broken Stone Pit is a possible source of some chemicals.

2. A potential, unidentified source northwest of well UNA-5 may be responsible for the methylene chloride and trichloroethylene in that area.

By CDM:

1. Benzene, toluene, and ethylbenzene from the primary lagoon are attenuated by biodegradation and never reach the town wells. Contamination at the wells is due to the Massachusetts Stone Pit fuel storage tank.

2. GZA's detection of methylene chloride and chloroform is due to analytical error. CDM found no methylene chloride, chloroform. trichloroethylene, or chlorobenzene in any lagoon or well samples.

practices. CDM concluded that 1,1,1 trichloroethane in the wells came from river infiltration, although no contamination by this chemical was found in the river. Contamination of the wells from the primary and emergency lagoons appears small for this chemical when compared with that for the other chemicals. Given GZA's noncommittal position, and more importantly, because this chemical has lower estimated health risks than the others, it seems reasonable to omit trichloroethane as a significant risk at the wells due to the contamination by the lagoons.

Both GZA and CDM agree that 1,1-dichloroethylene dumped in the primary and emergency lagoons is causing current contamination in the town wells.

CDM's position that benzene, toluene, and ethylbenzene plumes are attenuated by biodegradation is the subject of professional dispute. Dr. Saul A. Slapikoff, Associate Professor of Biology at Tufts University stated that the conditions necessary for biodegradation are not present in the aquifer (personal communication, 1981). Therefore, the lagoons cannot be eliminated as current or potential sources of contamination at the wells. However, both CDM and GZA recognize the fuel storage tank in the Broken Stone Pit as a possible source of these chemicals as well.

CDM maintained that GZA's analyses of methylene chloride and chloroform were erroneous and that these chemicals do not exist in the aquifer. However, the methylene chloride and chloroform must be considered in this risk analysis. GZA's sampling program was more extensive than CDM's; more samples were collected at more wells. Furthermore, GZA sampled the bottoms of the lagoons

while CDM collected only surface samples. GZA's plume maps for these two compounds show migration from the lagoons to the town wells. This evidence is strong enough to consider the lagoons to be the source of well contamination.

On the weight of this evidence, we conclude that the lagoons appear to be the source of 1,1 dichloroethylene, chloroform, methylene chloride, benzene, ethylbenzene, and toluene contamination at the town wells. Trichloroethylene contamination is primarily a result of induced infiltration from the Assabet River. 1,1,1 Trichloroethane presents a smaller hazard than the other chemicals, and its source is unknown.

APPENDIX D
GROUNDWATER TRANSPORT: FAST OR SLOW?

D.W. Cooper

Introduction

According to the estimates presented in the body of this report, the number of expected fatalities differs by about a factor of ten, depending on whether the concentrations in the Assabet wells No. 1 and No. 2 in the years after 1979 are substantially higher than the concentrations measured in 1979. These estimates are based on the assumptions of continued production at the company site and continued use of the wells. This appendix discusses the transport question in somewhat greater detail than did the main text.

Model

The model for the flow of the chemical components presented in Figure 4 of the text is for a plug flow of liquid through a column of soil. The ratio of the mean velocity of the chemical species, v_c, to the mean flow velocity, $\bar{v}$, is given by the following equation (Freeze and Cherry, 1979):

$$\bar{v}_c/\bar{v} = 1/(1 + p_b K_d/n) \qquad (D.1)$$

where p_b is the bulk density of the material and n is the porosity. K_d, the distribution coefficient, is defined as the mass of solute on the solid phase per mass of solid phase divided by the concentration of solute in solution; K_d has units of

volume per mass. K_d can be shown to be related to the partition coefficient, K_{om}, by the following expression:

$$p_b K_d/n = K_{om}(1-n)/n \qquad (D.2)$$

K_{om} is the ratio of the concentration in organic matter to the concentration in groundwater. For a porosity of 0.4 and a p_b of 2.0 g/cm^3, which are fairly typical values (see Freeze and Cherry, 1979), the retardation of velocity is equal to a factor of about 1/(1 + 1.5K). K is a partition coefficient for the ground as an entirety, but actually the organics are almost exclusively taken up by organic matter ("om") in the ground. The partition coefficient for the ground becomes

$$K = fK_{om} \qquad (D.3)$$

where f is the fraction (by volume) of the solid soil that is organic matter.

A correlation developed by Chiou et al. (Chiou, 1981) is:

$$K_{om} = 1.02 \times 10^4/S^{0.557} \qquad (D.4)$$

which had a correlation coefficient of r = 0.994 for 15 organic compounds (in a log-log regression); S, solubility, was in units of umole/liter.

Table D-1 shows our calculations of the partition coefficients K_{om}, using the correlation of Chiou et al. The first column has the chemicals of interest. The second gives the solubilities in ppm (generally from Hwang, 1982). the third shows the equivalent values of S in umole per liter. The last column gives our calculated values of K_{om}. These values are

Table D-1. Estimates of Partition Coefficients

Chemical	S^b (ppm)	S x 10^4 (umole/L)	K_{om}^c
1,1 dichloroethylene	5000	5.2	24
methylene chloride	16700	20.0	11
chloroform	9600	8.1	18
trichloroethylene	1100	0.84	65
ethylbenzene	206	0.19	147
benzene	1780	2.3	37
chlorobenzene		0.43[a]	94
toluene	535	0.58	80
1,1,1 trichloroethane		1.02[a]	100[a]
1,1,1 trichloroethane	5497	4.1	27

a. From Chiou (1981)
b. Hwang (1982)
c. $(g/L)_{om} / (g/L)_w$; w = water; om = organic matter 1.724 organic carbon), from Chiou (1981).

large enough that if the fraction of organics were large, the chemicals would be greatly slowed in their passage to the wells, in comparison with the velocity of water, and the situation would approximate our "slow transport" limit.

The uptake of the non-ionic organic chemicals, the type we are concerned with here, depends on the organic content of the soil itself; the uptake is proportional to the organic content for soils with the same particle size characteristics (Chiou, 1981). Although we were not able to find estimates for the organic matter composition of the ground in the aquifer, we have the following expert opinion from John Ayres of Goldberg, Zoino & Associates, consultants to the town and EPA: below the root zone and exclusive of any deposits of natural organic soils, the organic content of the Sinking Pond outwash aquifer will be essentially zero (Ayres, 1982).

This analysis favors adopting the "fast transport" assumption. Also in favor of the fast transport model is that the emissions from production on the company site have been flowing in the general direction of the Assabet River since the 1950s, when production commenced, and thus to the Assabet wells No. 1 and No. 2. The rate of water flow is such that the aquifer is traversed in about half a dozen years; this rate of transfer means that the concentrations in the general region of the wells should approach equilibrium values in 15 to 20 years. The town wells were operating from 1970 through the next decade, giving the concentrations a significant amount of time to approach equilibrium by the time sampling was performed. These

considerations argue against the hypothesis that much larger concentrations would be likely in the future.

However, the strong concentration gradients found moving out from the lagoons seem to favor the concept that adsorption is an important component of transport, supporting our slow transport hypothesis.

Factors other than adsorption that could also contribute to the strong concentration gradients along the flow path are:

1. longitudinal dispersion resulting from mixing and flow at different velocities at different depths;

2. lateral dispersion;

3. increasing dilution flow as the material moved away from the lagoons, along flow paths to which clean water was being added by natural recharge.

The above considerations lead us to conclude that the "fast transport" assumption is more likely to be true than the "slow transport" assumption, and that the lower estimates of expected fatalities, based on the "fast transport" assumption, are more likely to be correct than the higher estimates.

APPENDIX E
AIR POLLUTION FROM LAGOONS

D.W. Cooper

Introduction

The chemicals in the lagoons tend to evaporate from the water in which they are dissolved. Here we estimate both the rate of such evaporation for the chemicals being studied and the air pollution levels such evaporation could cause.

Evaporation

The materials dissolved in water will have vapor pressures, actually fugacites, that are much lower than if the materials were in their pure, unmixed state. The rate at which the materials are emitted from the surface depends upon transport from the bulk of the water mass to a narrow region ("film") near the lagoon's surface, transport through this film and through a gas film, and then into the bulk of the air.

Assuming there is no such material in the air before reaching the lagoons, the gas flux will be the fugacity of the material in the water divided by the total resistance to transport (Mackay, 1981):

$$N = HC/R_T \qquad (E.1)$$

where N is the flux, in moles per m2 per second. HC, the fugacity (atm), is the product of the Henry's Law constant (atm/mol/m3) and the species' concentration in the liquid (C,

mol/m^3). R_T is the total resistance to transport, and has the units of atm m^2 sec/mol. The resistance of the bulk fluid will be ignored, somewhat overestimating the emission rate. The resistance of the fluid film is equal to (Mackay, 1981):

$$R_L = H/K_L \qquad (E.2)$$

And the resistance of the gas film is given by:

$$R_G = RT/K_G \qquad (E.3)$$

where R is the gas constant (8.2 x 10^{-5} atm / m^3-mole- K) and T is the absolute temperature (K). Values for KL in the environment are typically from 10^{-4} to 10^{-5} m/s and for K_G are 3 x 10^{-3} to 3 x 10^{-2} m/s (Mackay, 1981).

Choosing high values of K_G and K_L produces low estimates of the resistance and thus high values for the flux. When the Henry's Law coefficients are larger than about 10^{-3} atm-m^3/mole, as they are for the chemicals we are interested in, the resistance of the gas phase is less than that of the liquid film. Neglecting the gas film resistance (thus overestimating flux) gives the very simple approximation:

$$N < HC/(H/K_L) = CK_L \qquad (E.4)$$

which indicates that the flux is somewhat less than the concentration of the material (moles/m^3) multiplied by the factor K_L (m/s).

Once an estimate of the flux is obtained, it can be converted into a mass emission rate (g/s) by multiplying it bythe surface area of the lagoon, A (m^2), and by the molecular weight of the material (g/mole).

Another simple estimate can be obtained by using the method proposed by Smith et al. (1980) for obtaining the coefficient k to be used in the following approximate equation:

$$dC / dt = -kC \qquad (E.5)$$

where k is the reciprocal of a time constant and C is the concentration in the lagoon, as before. A mass rate can be estimated by multiplying this equation by the volume of the lagoon, V(m).

A correction factor from Smith et al. (1980) recommended to apply to k, which is appropriate for oxygen, is between 0.5 and 0.9 for the chemicals we are studying. Using the k for oxygen for ponds, k = 0.1 to 0.2 per day (Smith et al., 1980), and 86,400 as the number of seconds in 24 hours, we have:

$$\dot{m} = CV(0.2/86,400) \qquad (E.6)$$

This is a semi-empirical estimate of the mass rate of emissions, $\dot{m}$.

The most elaborate methodology we employed was that presented by Hwang (1982). Both the gas film resistance and the liquid film resistance are estimated, from data for oxygen in water and water in air. The total resistance is obtained by summing these two. This methodology is inherently more accurate than either of the two methods above.

Transport

The mean concentration at a distance r from the site can be shown to be inversely proportional to $r^{1.5}$, for methodology in the Boston area, averaged over several years (Cooper,.1982). The same calculations show that the concentration expected a kilometer (km) away from a point source emitting 1 g/s would average 1 mg/m^3. Actually, the concentrations from an area source such as pond would be appreciably less.

Estimated Concentrations

Table E-1 shows our estimates of the concentrations averaged over a year at 1 kilometer (0.6 mile) from the lagoons for five chemicals found in the lagoons. The first three columns contain the approximations made using the methods of Smith et al. (1980), the Hwang (1982) approach, and the upper estimates provided by our first equation. The last column shows the industrial hygiene threshold limit values (TLV's), appropriate for an industrial population working a 40-hour week. Although limits for the population at large would be smaller because of the heterogeneity (following Hwang) are all more than 100 times less than the TLV's; therefore, we conclude that the air pollution contribution to risk from the lagoons has been negligible.

Table E-1. Estimates of Air Pollution Concentrations from Lagoons, Compared with Occupational Threshold Limit Values

Chemical	Air contamination estimates (mg/m^3)			Threshold limit values[a] (mg/m^3)
	Method of Smith et a1.(1980)	Method of Hwang (1982)	Eq. E.1	
1,1 dichloro-ethylene	0.043[a] 0.003[b]	0.14 0.15	<1 <1	40
methylene chloride	0.007 0.002	0.024 0.088	<0.2 <0.7	105
trichloro-ethylene	0.040 0.005	0.11 0.19	<1 <2	535
chloroform	0.002 0.0002	0.006 0.07	<0.05 <0.07	50
ethylbenzene	0.132 0.036	0.40 1.52	<3 <13	435

[a]Primary lagoon.
[b]Secondary lagoon.

APPENDIX F
HEALTH RISK ASSESSMENT METHODOLOGY

L.A. Beyer

For each of the nine pollutants found at the site, we made risk estimates for carcinogenicity, teratogenicity, reproductive toxicity, neurobehavioral toxicity, hepatotoxicity, renal toxicity, and other toxic effects. The basis and limitations of the cancer risk estimates are presented first, followed by a discussion of the method used to estimate the risks associated with the other six categories of health effects.

Cancer Risk

Table F-1 shows the cancer risk estimates for the nine toxins found in the waste site. To the best of our knowledge, these estimates include all of the most important estimates that have been calculated for these chemicals. Thus, we did not choose any particular model that has been used to estimate cancer risk over any other. The models are divided into two types: (1) a weight to weight (mg/kg/day) conversion from animals to humans, and (2) a surface area conversion. The scientific basis for these conversions are discussed later, but a cursory look at the table shows that the mg/kg/day conversion tends to give lower risk estimates than the surface area conversion.

Table F.1 Fatality Risk Estimates

Cancer Risk	Models Using Wt. to Wt. Conversion				Multistage Model Using Surface Area Conversion							
	Crouch & Wilson		Clement Arithmetic $\bar{x}$		EPA Ambient Water Quality Criteria				CAG		CEQ	NAS
	q^* $(mg/kg/day)^{-1}$	Lifetime Risk per 1 µg/l	potency $(mg/kg/day)^{-1}$	Lifetime Risk per 1 µg/l	Level Corresponding to Lifetime Risk of 10^{-5} Water & Fish µg/l	Fish Only µg/l	q^* $(mg/kg/day)^{-1}$	Lifetime Risk Per 1 µg/l	q^* $(mg/kg/day)^{-1}$	Lifetime Risk Per 1 µg/l	Lifetime Risk Per 1 µg/l	Lifetime Risk Per 1 µg/l
Benzene	10^{-3}	2.86×10^{-8}	1.64×10^{-3}	4.69×10^{-8}	6.6	400	5.29×10^{-2}	1.51×10^{-6}	6.8×10^{-1}	1.94×10^{-5}	4.4×10^{-6}	nd
Chloroform	10^{-2}	2.86×10^{-7}	3.79×10^{-3}	1.08×10^{-7}	1.9	157	1.83×10^{-1}	5.23×10^{-6}	1.67×10^{-1}	4.76×10^{-6}	4.1×10^{-6}	4.4×10^{-6}
1,1 Dichloro-ethylene (Vinylidene)			4.7×10^{-3}	1.34×10^{-7}	.33	18.5	1.04	2.97×10^{-5}	RD			nr
Ethyl Benzene					TOX	TOX			nd			nr
Methylene Chloride									nd			nd
Monochloro-benzene					TOX	TOX			nd			nd
Toluene					TOX	TOX			nd			nd
1,1,1 Tri-chloroethane	1.7×10^{-5}	4.86×10^{-10}			TOX	TOX			nd			nr
Trichloro-ethylene	7×10^{-4}	2×10^{-8}	9.6×10^{-3}	2.75×10^{-7}	27	807	1.26×10^{-2}	3.6×10^{-7}	1.26×10^{-2}	3.6×10^{-7}	3×10^{-7}	3.2×10^{-7}

q^* = slope of potency for human adults; to convert q^* to Lifetime Risk multiply $q^* \times 2.86\times10^{-5}$ mg/kg/day, which is the dose d = 2 l/day x 10^{-3} mg/l x 1/70 kg.
RD = CAG Redoing estimate
nr = not reviewed
nd = reviewing group feels there is not enough data on which to base a point estimate
TOX = only acute toxicity evaluated

The models used to estimate cancer risk were developed by E. Crouch and R. Wilson, Clements Associates, and K. Crump. All of these models use animal data, in a very few cases supplemented by human data, to predict the risk of cancer in human populations. Many problems arise when using animal data to predict human health effects, not the least of which is the lack of quality and consistency of the experimental data, a problem that has been compounded by incomplete reporting of the data. The National Cancer Institute's (NCI's) bioassay program, on whose data Crouch and Wilson base their estimates, has reduced experimental inconsistency to a minimum by imposing strict regulations on the program (Sontag, 1977). Because NCI data are generated under consistent conditions, they are internally comparable: however, the comparability of NCI's data with other experimental conditions is still difficult to ascertain.

Another problem in using animal data to predict human health effects is that carcinogenesis assays are statistically significant only with cancer incidences of 5 to 10% higher. To achieve these incidences, substances have to be administered in high doses, often throughout a large portion of an animal's lifetime. To estimate the risks to humans of doses that are typically much lower than those used in the animal experiments, two extrapolations are required: one is from the incidence found at high doses to an estimated incidence at low doses; the second is from experimental animals to humans. The bases for both of

these extrapolations are subjects of active scientific debate, and the lack of data to substantiate many of the assumptions made in the process of extrapolation introduces a high degree of uncertainty in the risk estimates made.

In extrapolating from high to low doses, a mathematical relationship between dose and effect must be assumed. Many such models have been proposed, including the linear, multistage, one-hit, probit, and logit-probit models. Because all of these models appear to fit high-dose data equally as well, there is no statistical way to choose the model that will most accurately predict health effects at low doses. Even for toxins for which relatively good data exist (such as aflatoxin and ionizing radiation), no model consistently predicts the high-dose risk estimate better than any other (Carlborg, 1979; NAS, 1977).

To extrapolate from high doses to low doses, the conventional method has been to use the model that has the best match with our current understanding of the biological genesis of cancer. In the judgment of many experts, linear extrapolation at the low dose region of the dose-reponse curve is the most prudent estimation method. It is mathematically simple and much, though not all, experimental and epidemiological evidence is in its favor, both for radiation and chemical carcinogenesis (Mantel and Schneiderman, 1975, and Hoel et al., 1975, as cited in Weinhouse, 1977). The possibility of strongly non-linear responses, including virtual thresholds, has not been ruled out in all cases.

All of the models whose risk estimates are used in this report are based on this type of extrapolation, with the exception of Clements Associates' estimates. The Clements Associates' estimates are based on arithmetic means, which when used to estimate the risk to a population assume a strictly linear relationship, not a linear extrapolation at the low-dose region of a dose-response curve. Theoretically, linear models produce the most conservative risk estimates, avoiding underestimates at the cost of possible overestimates, assuming the same data are used. However, because Clements Associates averaged data from many experiments and the other models used data from one carefully chosen experiment, the results are not strictly comparable. As a result, the Clements Associates' estimates were among the least conservative.

The issue of comparability is also complicated by the manner in which the various models convert animal dosages to human dosages. This animal-to-people conversion is usually done on a mg/kg/day basis or by surface area. The mg/kg/day conversion implies that, per kg of body weight, the exposure to a milligram of toxin is the same in the experimental animals and humans. But, this may not be true. Blood circulates approximately 20 times as rapidly in a mouse as in a man, so that the time a substance spends in the plasma (excluding metabolism) is longer in a larger mammal than in a smaller one. Thus, for the same milligram per kilogram dose, human tissues are exposed to a substance for a longer time than mouse tissues. This is consistent with data obtained in studies of anticancer drugs that showed, on a milligram-per-kilogram basis, a mouse required 12

times as much drug to respond as did a human, a rat 6 times as much, and a monkey and dog 2 to 3 times as much. When the data were expressed by surface area, on a milligram-per-square-meter basis, the differences between species were sharply reduced (Friereich, 1966). However, other calculations by Crump and Howe (1980) and Crouch and Wilson (1979) show that the mg/kg/day conversion procedure gives the best correlation between carcinogenic potencies in animals and humans.

Other differences between humans and rodents also complicate the extrapolation of data from one to the other. The cell division rate is greater in small animals than in humans. In mice, for example, gut or marrow cells' cycle time is about half that of comparable cells in humans. Also, a human's life span is 35 times that of a mouse. In an average lifetime, a human would have 70 times as much cell regeneration as a mouse, so that humans have a greater chance for a DNA mutation to be expressed.

Excretion and reabsorption, distribution and storage, metabolism, differences in receptor sites, and differences in absorption can also vary between species and within species. Although within-species variability is minimized in laboratory experiments, humans are not genetically homogeneous. In fact, genetic differences can cause the plasma concentration of substances given in equal doses to vary by a factor of 100 (Weber, 1976, as cited in NAS, 1977). Also, humans live in various environmental conditions, eating a great variety of food and experiencing considerable variation in the intake of, or exposure to, environmental pollutants.

Potentiation, antagonism, and synergism are also important factors in risk analysis. Many unexpected toxic reactions to therapeutic agents have been caused by the interactions of compounds that were safe when given alone (Conney and Burns, 1972 as cited in NAS, 1977). Chemicals found in the environment have also been found to act synergistically, such as cigarette smoke and asbestos (Selikoff et al., 1968, as cited in Crump and Guess, 1980). Antagonism has also been reported in the literature (Smyth et al., 1969). Few studies have been performed to investigate interactive effects in low-dose chronic experiments. Smyth's study concluded that, after testing 27 industrial chemicals in all possible pairs, when the synergistic and antagonistic effects were averaged, the net effect was that of additivity, which satisfactorily predicted the toxicity of a large population of the pairs. Based on this evidence and the American Conference of Governmental Industrial Hygienists' policy, now codified and enforced by the Occupational Safety and Health Administration (OSHA), we too treat the cancer risk of various toxins as additive.

In spite of the many limitations and problems inherent in using animal data to predict human cancer risk, it has, in the past, been very valuable in this regard (Tomatis, 1977). Furthermore, since experiments cannot be ethically performed on humans and epidemiological studies are difficult, expensive, time-consuming, often ambiguous, and in some cases impossible to perform, animal data are the only dose-response data available on which to base a risk assessment analysis.

Risk Estimate Models for Carcinogenicity

Crouch and Wilson's model is a no-threshold model, which is assumed to be linear in the low dose region of the dose-response curve. The model uses as the relevant measure of dose the ratio of the weight of carcinogen to the body weight of the animal, where the lifetime probability of an animal getting cancer, P, depends on the integrated lifetime dose of the carcinogen. At high doses it is assumed that the dose-response curve saturates to one exponentially and that the number of animals getting cancer is binomially distributed with probability P.

The model has the form:

$$P = 1 - (1 - \alpha) \exp\left(- \frac{\beta d}{1-\alpha} \right) \qquad \text{(F.1)}$$

At low doses (d), this equation reduces to P = a + Bd, where $\alpha \geq 0$, $\beta \geq 0$, and $d < (1-\alpha/\beta)$. a and B are estimated from NCI bioassay data, using maximum likelihood techniques. (See Crouch and Wilson, 1979 for a more complete description.) The results of this model are reported in terms of potency, β (the linear slope of the low-dose portion of the dose-response curve) in units of $(mg/kg/day)^{-1}$. The estimates cited in this report are from Crouch and Wilson, 1979 and personal communication with E. Crouch. To convert to lifetime risk, we assumed (as did Crouch and Wilson) a consumption of 2L/day of water (containing 1 ug/L

or 1 ppb of toxin) by a 70-kg adult. This leads to a conversion factor of 2.86×10^{-5}:

$$\frac{(2L/day)(10^{-3}mg/L)}{70\ kg} = 2.86 \times 10^{-5}(mg/kg/day)$$

The Clements Associates' risk estimates are derived from an averaging of doses in animal experiments, resulting in a linear model. These doses (Clements Associates, 1982) are reported in terms of potency, $(mg/kg/day)^{-1}$. We multiplied them by the conversion factor developed above, 2.86×10^{-5} mg/kg/day. to get lifetime risk estimates.

The other models used are all based on the linear, no-threshold multistage dose-response model developed by Dr. Kenneth S. Crump. Grump's model has been computerized by the EPA and is called GLOBAL 79. The model estimates P(d), the lifetime risk (probability) of cancer at dose d, using the following equation:

$$P(d) = 1-\exp[-(q_0+q_1d^1+q_2d^2+\ldots+q_kd^k)] \qquad (F.2)$$

where q_0 equals the background cancer rate, $q_i \geq 0$, and i-0,1,2,...,k. The equation is solved for q_1 which is the slope of the sigmoidal dose-response curve at low doses. At low doses, upper and lower 95% confidence limits on the dose are determined from the upper 95% confidence limit around q_1, q_1*. The upper limit, q_1*, is calculated by increasing q_1 to a value q_1* such that the log-likelihood is maximized, assuming a chi-square distribution. It is assumed that the milligram per surface area dose is equivalent between species. The carcinogen potency factor, q*, is the slope of the 95% confidence interval at low

doses and has units of $(mg/kg/day)^{-1}$. Once determined, q* is multiplied by the conversion factor of 2.86×10^{-5} mg/kg/day to determine the risk from lifetime continuous exposure at 1 ppb. (See U.S.E.P.A., 1980, for a more detailed description.)

Three different groups use Crump's model to calculate lifetime risk. They are the Cancer Assessment Group (CAG), the Council on Environmental Quality (CEQ), and the National Academy of Sciences (NAS). CAG has provided risk estimates for the EPA Ambient Water Quality Criteria as well as for other EPA programs.

The EPA Ambient Water Quality Criteria is in the November 28, 1980 Federal Register:. In arriving at these criteria, q* was determined from GLOBAL 79, and P, the probability of cancer, was assigned. The allowable concentration, C, in mg/L, was calculated as follows:

$$C = \frac{70P}{q^*(2+.0065R)} \qquad (F.3)$$

where:

70 = weight of an adult, kg
P = probability of cancer
q* = potency, $(mg/kg/day)^{-1}$
2 = the average amount of water drunk by an adult per day, L
R = the bioaccumulation factor, L/kg of fish
0.0065 = the average amount of fish eaten by an adult per day, kg/day

Rearranging this equation we obtain:

$$q^* = \frac{70P}{C(2+.0065R)}$$

The Ambient Water Quality Criteria Documents do not give risk estimates for water consumption only. That is, they give risk estimates assuming a fish consumption of 0.0065 kg per adult per day and risk estimates for consumption of 2 liters of water and 0.0065 kg fish per day. from these estimates, q* can be calculated using two equations with two unknowns (q* and R).

The CAG risk estimates were found in the work by Clements Associates (1982). The CAG office of the EPA was contacted to ensure that there were no other risk estimates applicable to this study. The CAG estimates are given as q*, $(mg/kg/day)^{-1}$; we converted them to lifetime risk estimates by multiplying them by 2.86×10^{-5} mg/kg/day (as indicated in Table F-1).

The CEQ estimates can be found in the material presented by Crump and Guess (1980). These estimates are reported as upper 95% confidence interval lifetime risk estimates, in ug/L. No conversion was needed.

The NAS estimates can be found in the report by NAS (Volume 1, 1977). These estimates were given as lifetime risk estimates, assuming a consumption of one liter of water per day. To make these estimates consistent with other models, which assume two liters per day of water consumption, we doubled the rates.

Although one might expect that risk estimates based on the same model should give identical results for the same substances, this does not always hold true. The reason for the inconsistencies found in Table F-1 is that the most recent data

available were used in calculating each estimate. Since the estimates were made at different times, different data were sometimes used in making them.

Toxicity-Estimates

The risk estimates for the toxic effects of teratogenicity, reproductive toxicity, renal toxicity, neurobehavioral toxicity, and other toxic effects are shown in Table F-2. These estimates are based on a linear model proposed by Clements Associates (1981), where P is the incidence per minimum effective dose (MED) times the actual human dose. Clements Associates estimated incidence per MED by surveying both primary and secondary literature, extracting the dose response data, and then calculating the arithmetic mean, the geometric mean, and the standard deviation of the logarithms. Because of the paucity of data on toxic effects, the conservative estimate that a linear model makes was used: we used the arithmetic means as the bases for our estimations. Less confidence can be placed in these estimate than in the fatality estimates.

Note, however, that toxic effects other than carcinogenicity may also exhibit a no-threshold, sigmoidal dose-response curve; the sigmoidal model, instead of representing a dichotomous. response, represents the cumulative frequency distribution of organisms showing an effect as a function of dose (Pfitzer and Vouk, 1979). As the data and our understanding of toxic effects improve, it may become appropriate to use a multistage model to estimate the incidence of cumulative toxic effects instead of the linear model used in this analysis.

Table F.2 Illness Risk Estimates

	Reproductive Toxicity		Teratogenicity		Hepatotoxicity		Renal Toxicity		Neurobehavioral Toxicity		Other Toxicity	
	potency $(mg/kg/day)^{-1}$	Lifetime Risk	potency $(mg/kg/day)^{-1}$	Lifetime Risk	potency $(mg/kg/day)^{-1}$	Lifetime Risk	potency $(mg/kg/day)^{-1}$	Lifetime Risk	potency $(mg/kg/day)^{-1}$	Lifetime Risk	potency $(mg/kg/day)^{-1}$	Lifetime Risk
Benzene	6.17×10^{-3}	1.76×10^{-7}	4.53×10^{-4} T	1.3×10^{-8}	nd	-	nd	-	7.57×10^{-3}	2.17×10^{-7}	4.45×10^{-3}	1.27×10^{-7}
Chloroform	4.08×10^{-3}	1.17×10^{-7}	3.45×10^{-3}	9.87×10^{-8}	3.24×10^{-2}	9.27×10^{-7}	3.25×10^{-2}	9.3×10^{-7}	8.0×10^{-4}	2.29×10^{-8}	9.5×10^{-3}	2.72×10^{-7}
1,1 Dichloro-ethylene (Vinylidene)	nd	-	nd	-	5.54×10^{-3}	1.58×10^{-7}	nd	-	nd	-	nd	-
Ethyl Benzene	nd	-	nd	-	nd	-	nd	-	nd	-	nd	-
Methylene Chloride	nd	-	nd	-	4.09×10^{-4}	1.17×10^{-8}	1.09×10^{-3}	3.12×10^{-8}	3.6×10^{-3}	1.03×10^{-7}	3.85×10^{-4}	1.1×10^{-8}
Monochloro-benzene	nd	-	nd	-	nd	-	nd	-	nd	-	nd	-
Toluene	2.58×10^{-4}	7.38×10^{-9}	nd	-	1.21×10^{-4}	3.46×10^{-9}	nd	-	1.13×10^{-2}	3.23×10^{-7}	nd	-
1,1,1 Tri-chloroethane	5.72×10^{-5}	1.64×10^{-9}	nd	-	7.99×10^{-4}	2.29×10^{-8}	nd	-	8.03×10^{-4}	2.3×10^{-8}	3.69×10^{-4}	1.06×10^{-8}
Trichloro-ethylene	nd	-	nd	-	5.28×10^{-4}	1.51×10^{-8}	1.82×10^{-4}	5.21×10^{-9}	9.41×10^{-2}	2.69×10^{-6}	.155	4.43×10^{-6}

potency = arithmetic mean from Clements Assoc.
Lifetime Risk = potency x 2.86×10^{-5} mg/kg/day
T = Clements TIP Score
nd = no data

Clements Associates' data were obtained from the work by them (Clements Associates, 1982). The scores (incidence/MED) are presented in $(mg/kg/day)^{-1}$, which we converted to lifetime risk by multiplying by 2.86×10^{-5} mg/kg/day. In as much as the Clements Associates' data are based on extrapolations from animal studies, the interpretation of the data suffers from the same limitations as the cancer risk estimates.

REFERENCES

Ayres, John of Goldberg, Zoino and Associates, Newton, MA. September 1982. Personal communication.

Camp, Dresser & McKee Inc., Boston, MA. April 1980. W.R. Grace & co. Organic Chemicals Division Review of Report on Groundwater Contamination at the Assabet Well Field, Acton, Massachusetts.

Camp, Dresser & McKee Inc., Boston, MA. August 1982. W.R. Grace & co. Acton Environmental Program Report on Sampling and Analysis Program, Volume I.

Carlborg, F.W. 1979. Cancer, mathematical models, and aflatoxin. Food Cosmet. Toxicol., 17:159-166.

Caswell, W.B. 1979. Ground Water Handbook for the State of Maine. Maine Geological Survey, Maine.

Chiou, C. 1981. Partition coefficient and water solubility in environmental chemistry. In Saxena, J. and Fisher, F. eds. Hazard Assessment of Chemicals, Current Developments, Volume I. New York: Academic Press.

Clements Associates, Inc., Arlington, VA. August 15, 1981. Toxics Integration Program Scoring of Selected Pollutants for Relative Risk - Appendix C in An Industry Approach for the Regulation of Toxic Pollutants. Prepared by Putnam, Hayes, and Bartlett, Inc., Cambridge, MA, for Toxics Integration Project [now known as the Program Integration Project (PIP)], U.S. Environmental Protection Agency.

Clements Associates, Inc., Arlington, VA. July 16, 1982. Health Score Improvement Task 1: Data Development. Draft Report prepared for American Management Systems, Inc.

Cooper, D.W. October 1982. Receptor-oriented Source-receptor Analysis. Presented at Air Pollution Control Association Specialty Meeting on Receptor Models Applied to Contemporary Problems, Danvers, MA.

Crouch, E. and Wilson, R. 1979. Interspecies comparison of carcinogenic potency. Journal of Toxicology and Environmental Health, 5:1095-1118.

Crump, K.S. and Guess, H.A. December 1980. Drinking Water and Cancer: Review of Recent Finding and Assessment of Risks; Prepared by Science Research Systems, Inc., Ruston, LA, for the Council on Environmental Quality, NTIS Number PB81-128167.

Crump, K.S. and Howe, R.B. August 1980. Approaches to Carcinogenic, Mutagenic and Teratogenic Risk Assessment. Prepared by Science Research Systems, Ruston, LA, for Meta Systems, Inc., Cambridge, MA. Task A, Subtask No. 5, Summary Report, Contract No. 68-01-5975, U.S. Environmental Protection Agency.

Department of Urban & Environmental Policy, Tufts University, Medford, MA. June 1981. Chemical Contamination-of Water: The Case of Acton, Massachusetts.

Freeze, R.A. and Cherry, J.A. 1979. Groundwater. New Jersey: Prentice-Hall, Inc.

Frierich, E.J. et al. 1966. Quantitative comparison of toxicity of anti-cancer agents in mouse, rat, hamster, dog, monkey, and man. Cancer Chemotherapy Reports, 50(4):219-244.

Gardner, J.J. and Ayres, J. 1980. Acton, Massachusetts: A Groundwater Contamination Problem. Proceedings of the 1980 Hazardous Waste Spill Conference, Vanderbilt University, Louisville, Kentucky.

Goldberg, Zoino & Associates, Inc., Newton, MA. January 1980. Groundwater Investigation - Assabet Well Field, Acton, Massachusetts, Volumes I and II.

Goldberg, Zoino & Associates, Inc., Newton, MA. April 1982. Disposal Area Characterization - W.R. Grace & Company Property.

Hwang, S.T. February 1982. Toxic emissions from land disposal facilities. Env. Progress, 1(1):46-52.

Lycott Environmental Research, Inc., Southbridge, MA. August 18, 1981. Final Report, Ground-Water Resources Evaluation and Aquifer Protection Plan, Phase II, for the Town of Acton, Massachusetts.

Mackay, D. 1981. Environmental and laboratory rates of volitilization of toxic chemicals from water. In Saxena, J. and Fisher, F. eds. Hazard Assessment of Chemicals, Current Developments Volume I. New York: Academic Press.

National Academy of Sciences. 1977. Drinking Water and Health, Vol. 1. Washington, D.C.: National Academy Press.

National Academy of Sciences. 1980. Drinking Water and Health, Vol. 3. Washington, D.C.: National Academy Press.

Pfitzer, E. and Vouk, V. 1979. Mathematical and statistical aspects of dose-effect and dose-response relationships. In Friberg, L. et al., eds. Handbook on Toxicology of Metals. Elsevier/North-Holland: Biomedical Press.

Smith, J.H. et al. November 1980. Prediction of the volatilization rates of high-volatility chemicals from natural water bodies. Env. Sci. & Tech., 14(11):1332-1337.

Smyth, H., Weil, C., West, J., and Carpenter, C. 1969. An exploration of joint toxic action: twenty-seven industrial chemicals intubated in rats in all possible pairs. Toxicology and Applied Pharmacology, 14:340-347.

Sontag, J.M. 1977. Aspects in carcinogen bioassay. In Hiatt, H. et al., eds. Origins of Human Cancer, Book C. Cold Spring Harbor: Cold Spring Harbor Laboratory.

Tomatis, L. 1977. The value of long-term testing for the implementation of primary prevention. In Hiatt, H. et al., eds. Origins of Human Cancer, Book C. Cold Spring Harbor: Cold Spring Harbor Laboratory.

U.S. Environmental Protection Agency. August 8, 1980. The Carcinogen Assessment Group's method for determining the unit risk estimate for air pollutants. Prepared by the Carcinogen Assessment Group in the Office of Health & Environmental Assessment.

U.S. Federal Register. Friday, November 28, 1980. 45:231:79323-79356.

Weinhouse, S. 1977. Problems in the assessment of human risk of carcinogenesis from chemicals. In Hiatt, H. et al., eds. Origins of Human Cancer, Book C. Cold Spring Harbor: Cold Spring Harbor Laboratory.

Zielhuis, R.L. April 1982. Standards for chemical quality of drinking water: a critical assessment. Int. Arch. Occ, & Env. Hlth., Vol 50.